



Fiscal composition, state capacity, and the production of longevity: Macro-micro evidence from Kenya

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ABSTRACT

Public health spending is central to human capital formation, yet the role of fiscal composition in shaping longevity in Sub-Saharan Africa remains underexplored. This paper investigates the intertemporal trade-offs of health financing in a low-income African setting, focusing on Kenya, using a dual empirical strategy: A Vector Error Correction Model (VECM) of national time-series data (1981-2023) and region fixed-effects models on census microdata. Macroeconomic results reveal a structural bias in fiscal composition with implications for long-run state capacity. The recurrent health expenditure ratio shows a significant negative long-run association with life expectancy ($\beta = -28.39, p < 0.05$). While recurrent spending improves short-run service delivery, it is capital accumulation through infrastructure, technology, and sanitation that shifts the longevity frontier. Micro-level analysis highlights distributional effects: recurrent spending interacts positively with maternal education ($\beta = 0.007, p < 0.05$) substituting for household human capital deficits and reducing child mortality among children of uneducated mothers. Female secondary education parity emerges as a binding macro constraint ($\beta = 250.71, p < 0.001$) suggesting diminishing returns to primary schooling. We also document a kinship dividend: higher dependency ratios protect child survival ($\beta = -0.389, p < 0.001$) challenging conventional dependency-burden models and underscoring the role of extended family systems. Taken together, these findings support a twin-track fiscal strategy: governments should safeguard capital health investments to build long-run state capacity, while deploying recurrent expenditure strategically to mitigate inequality across regions and households.

Contribution/Originality: This study contributes to the existing literature by examining how the composition of public health spending affects longevity in Kenya. This study uses a new estimation methodology combining VECM with micro-level fixed-effects. The paper's primary contribution is finding that spending composition matters more than total spending in LMIC settings.

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1. INTRODUCTION

Over the past four decades, Kenya has achieved substantial improvements in human longevity, with life expectancy rising from barely above 50 years in the early 1980s to over 66 years in the early 2020s (World Bank, 2023). These improvements mirror broader gains across Sub-Saharan Africa (SSA), where investments in health systems, education, and institutional reforms have contributed to notable reductions in mortality (Cutler, Deaton, & Lleras-Muney, 2006; Jamison et al., 2013). However, despite this progress, survival outcomes remain fragile and unevenly distributed. Kenya faces persistent pressures from demographic aging, fiscal constraints, and deep regional disparities (Kenya Ministry of Health, 2021). This divergence raises a fundamental economic question that extends beyond Kenya to the broader developing world as to whether it is the volume of public health spending that matters for survival, or the composition of that spending.

Economic development theory has long posited a direct link between public health spending and population longevity. Yet, the empirical record presents a confounding puzzle, which is that while aggregate health expenditures have risen significantly, improvements in life expectancy and child survival have often been nonlinear or stalling.

While according to Chansa (2025) political incentives in many developing economies often distort fiscal priorities favouring visible, capital-intensive development projects (e.g., hospital construction and equipment procurement) over recurrent expenditures essential for system functionality (such as staff remuneration, medicines, and maintenance), there remains limited empirical evidence on whether the composition of health spending between development and recurrent categories significantly influences population longevity. Existing studies document allocation patterns, but empirical links to long-term health outcomes are not yet well established.

Kenya provides an ideal laboratory to investigate this fiscal-demographic nexus. The country is undergoing a structural shift in its fiscal architecture, where health expenditure is now heavily recurrent-oriented, with nearly 75% of spending absorbed by wages and operations, potentially crowding out long-term system strengthening (Barasa, Rogo, Mwaura, & Chuma, 2018; Meheus & McIntyre, 2017). Concurrently, the country has implemented landmark reforms, including Free Primary Education (FPE) since 2003 (Duflo, 2001; Glennerster, Kremer, Mbiti, & Takavarasha, 2011) and the 2010 Devolution of health functions to forty-seven county governments (Cheeseman, Lynch, & Willis, 2016). Understanding how these specific fiscal and institutional shifts interact with demographic pressures such as high dependency ratios (Preston, 1975) to drive longevity is a blueprint for the efficient allocation of scarce public resources.

Despite the intuitive appeal of linking fiscal policy to health outcomes, the existing literature suffers from four critical gaps that this paper addresses. First, fiscal data often mask important distinctions because they are aggregated in ways that obscure underlying patterns. Most cross-country growth regressions treat health expenditure as a monolithic block (e.g., (Baldacci, Clements, Gupta, & Cui, 2008; Nixon & Ulmann, 2006)). By failing to disaggregate between recurrent (operational) and development (capital) spending, previous studies mask the distinct mechanisms through which these expenditures operate. Capital investment builds infrastructure that affects survival over generations, while recurrent spending provides the immediate liquidity (drugs, wages) required to save lives today. Empirical evidence on this distinction is extremely limited in low-and middle-income countries (LMICs) due to data scarcity (Anyangwe & Mtonga, 2007; Musah, Aawaar, Musah, & Abdul-Mumuni, 2025).

Second, there is limited integration of macro-fiscal trends with micro-demographic realities. Most studies either use national time-series data that overlook distributional heterogeneity (Makuta & O'Hare, 2015) or rely on micro-level household surveys (such as DHS) that treat the macroeconomic environment as fixed (Rutstein, 2000). As a result, we still lack evidence on how shifts in national budgets and fiscal volatility affect a child's chances of survival within a particular household, conditional on that household's characteristics.

Third, the interaction between fiscal policy and gender parity remains underexplored. While the *missing women* literature establishes gender inequality as a drag on development (Klasen, 2002; Sen, 1992), there is limited evidence about how this interacts with government spending. An important unanswered question is whether public health spending can substitute for maternal agency in patriarchal settings, or whether such spending only becomes effective when coupled with female empowerment.

Finally, the role of demographic pressure is often modelled linearly as a burden on growth (Bloom & Canning, 2000). In the African context, research rarely tests the kinship support hypothesis, which suggests that extended family structures can turn what appear to be high dependency ratios into a source of protection for child survival, complicating the predictions of conventional Western economic models.

In this paper, we fill these gaps by integrating macro-fiscal, demographic, and micro-census evidence to examine how the composition of health spending, demographic pressures, and gender parity jointly shape longevity in Kenya between 1981 and 2023. We construct a dataset merging national data from the Kenya National Bureau of Statistics (KNBS), the World Bank, and Penn World Tables to create an annual series of life expectancy, development-to-health and recurrent-to-health expenditure ratios, dependency ratio of the non-working to the working population, and institutional reform indicators. We then combine this with harmonized census microdata (IPUMS and KNBS), constructing region-specific measures of elderly shares and child survival.

The analysis proceeds in two stages. First, we estimate a Vector Error-Correction Model (VECM) to identify long-run cointegrating relationships between longevity and fiscal composition. This approach allows for rigorous distinctions between short-run shocks and long-run equilibrium, specifically testing the elasticity of life expectancy to recurrent versus development spending. Second, we bridge the macro-micro divide by estimating region-fixed effects panel regressions. We then link national fiscal and gender metrics to individual-level child survival outcomes derived from the census data. The identification strategy relies on the interaction of temporal variation in national policy with

spatial variation in household characteristics (specifically maternal education). This allows for the testing of substitution effects, whether high recurrent public spending reduces the survival disadvantage of children born to uneducated mothers.

This study makes four contributions to the African development and public finance literature. First, it provides rare long-run evidence that fiscal composition, rather than aggregate health spending, governs the longevity transition in a low-income economy. Second, it demonstrates that recurrent health expenditure functions primarily as a redistributive instrument, equalizing survival outcomes across households rather than shifting the national longevity frontier. Third, by explicitly modeling interactions between national fiscal policy and maternal education, the paper identifies a substitution mechanism whereby public health systems partially offset household human capital deficits. Finally, the findings challenge the conventional dependency-burden narrative by showing that extended family structures in Kenya operate as a protective health input, revealing an African kinship channel that standard demographic models overlook.

What this paper adds is not another confirmation that economic development and individual characteristics correlate with health outcomes. Rather, it identifies the mechanisms through which fiscal structure and household human capital interact to shape survival in a low-income African setting. Existing work typically treats public health expenditure as a single aggregate and implicitly assumes that its effects are monotonic and complementary to individual endowments. By contrast, this paper shows how governments spend matters as much as how much they spend, and that the effects of public spending are neither uniform nor always complementary. Using a forty-year cointegrated time-series framework, we demonstrate that capital-intensive health investment drives the long-run longevity frontier, while recurrent spending primarily redistributes survival chances within cohorts. Linking these macro-fiscal dynamics to census microdata further reveals a substitution effect: recurrent public health spending compensates for deficits in maternal education, reducing inequality in child survival rather than amplifying it. This macro-micro interaction, together with evidence that extended family structures reverse the conventional dependency burden in African households, offers new insights that cannot be recovered from either aggregate correlations or household-level analyses alone.

2. LITERATURE REVIEW

The relationship between fiscal policy and health outcomes is well established in development economics, yet important gaps remain in understanding the transmission mechanisms, especially in low-income settings. This section reviews five strands of literature: public expenditure quality, demographic pressure, gender inequality, institutional reforms, and macro-micro linkages, and highlights the specific theoretical and empirical gaps that this paper seeks to address.

Despite extensive research on the relationship between public spending, individual characteristics, and health outcomes, three limitations persist in the African context. First, most studies rely on static correlations or short-panel models that cannot distinguish long-run structural effects from short-run fiscal volatility. Second, public health expenditure is almost universally treated as an aggregate, masking the distinct intertemporal roles of capital accumulation and recurrent operational spending. Third, the literature overwhelmingly assumes complementarity between public inputs and household human capital, leaving untested the possibility that state capacity may substitute for maternal education in contexts of extreme deprivation. These limitations are particularly consequential in Sub-Saharan Africa, where fiscal rigidity, extended family systems, and uneven human capital accumulation challenge standard public finance assumptions. This paper addresses these gaps by combining cointegrated macro-fiscal analysis with census-based micro evidence to identify both the long-run production of longevity and the distributional mechanisms through which public spending reshapes survival.

2.1. Public Expenditure, Fiscal Composition, and Health Outcomes

A substantial body of literature examines the relationship between public health spending and aggregate health outcomes, often reporting positive but mixed effects. Cross-country analyses consistently show that increases in total government health spending reduce infant and maternal mortality, primarily by improving access to essential services and lowering financial barriers (Anyangwe & Mtonga, 2007; Bokhari, Gai, & Gottret, 2007; Kiross, Chojenta, Barker, & Loxton, 2020). These effects tend to be stronger in low-and middle-income countries (LMICs), where public spending represents a larger share of overall health financing (Moreno-Serra & Smith, 2012).

Yet a critical problem of aggregation bias remains. Most studies treat health expenditure as a single category, failing to distinguish between capital (development) and recurrent (operational) outlays (Fan & Savedoff, 2014). Development spending supports long-term investments such as hospitals and laboratories, while recurrent spending covers salaries, medicines, and routine operations of the health facilities. Chansa (2025) argues that the binding constraint on health service delivery is often not the absence of infrastructure but the rigidity of recurrent budgets, including wages for healthcare workers and funds for consumables.

In the African context, evidence on this distinction is emerging but still limited. Barasa et al. (2018) show that Kenya's public health system suffers from chronic under-investment in capital infrastructure, with development spending accounting for only 10–25 percent of total health expenditure. By contrast, studies from South Africa suggest that capital spending contributes disproportionately to long-run reductions in mortality (Ataguba & McIntyre, 2012). Despite these insights, almost no studies have applied Vector Error-Correction Models (VECM) to explicitly differentiate the short-run and long-run elasticities of recurrent versus development expenditure. This paper addresses

that gap by estimating an over 40-year cointegrated model to identify which component of fiscal policy drives the longevity transition.

2.2. Demographic Pressure: Burden or Buffer?

The literature on demographic structure is largely shaped by the “dependency burden” narrative. Classical transition theory argues that high ratios of non-working dependents to working-age populations divert resources from investment toward consumption, thereby straining fiscal capacity and household welfare (Bloom, Canning, Rosenberg, & Ghani, 2011; Lee & Mason, 2011). In Kenya, where nearly 40 percent of the population consists of dependents (KNBS, 2023), this demographic pressure is often assumed to crowd out health investments (Dyson, 2010).

In the context of African households, however, this linear view may be misleading. Maitra (2004) and others working on intra-household bargaining suggest that extended family structures frequently provide essential non-market services, particularly childcare and risk pooling, which remain invisible in standard economic models. A high dependency ratio may, in fact, signal the presence of grandmothers or older siblings who contribute critical caregiving labor, potentially improving child survival. Although Makuta and O’Hare (2015) explore this dynamic using cross-sectional data, few studies rigorously test the kinship support hypothesis against the resource strain model with long-run time series. This paper addresses this gap by incorporating dependency ratios into a VECM framework to assess their net impact on aggregate longevity.

2.3. Gender Parity as a Macroeconomic Constraint

The macroeconomic consequences of gender inequality have been rigorously examined by Klasen (2002) and Sen (1992). The mechanism is well established with the argument that limiting female education reduces the demographic dividend by sustaining high fertility and lowering child survival (Almond, Currie, & Duque, 2018; Duflo, 2012). In Kenya, the 2003 Free Primary Education (FPE) reform substantially narrowed gender gaps and strengthened female human capital formation (Bold, Kimenyi, Mwabu, Ng’ang’a, & Sandefur, 2018; Glennerster et al., 2011).

Despite these advances, an important gap remains concerning the level of education that is most consequential for health transitions. While gender disparities in primary schooling have largely closed, gaps at the secondary level persist. The literature explicitly linking secondary education gender inequality to aggregate life expectancy, rather than to economic growth alone, is limited. Moreover, little is known about how gender parity interacts with fiscal policy. One unresolved question is whether robust public health spending can substitute for maternal agency in patriarchal settings, or whether such spending is effective only when accompanied by female empowerment. This paper contributes rare empirical evidence to address this interaction.

2.4. Institutional Reforms: Devolution and Service Delivery

Another strand of literature examines the role of governance structures in shaping health outcomes. Kenya’s 2010 Constitution, which devolved health functions to 47 county governments, marked a major institutional reform. Early evaluations suggest that devolution enhanced accountability and strengthened localized planning (Cheeseman et al., 2016; Tsofa, Goodman, Gilson, & Molyneux, 2017), although concerns remain about rising wage bills and fragmented procurement systems.

Despite the scale of this reform, its broader demographic and macroeconomic impacts are not well understood. Existing studies tend to emphasize process indicators such as expenditure tracking, while paying less attention to long-term outcomes like changes in life expectancy. By treating devolution as a structural break within a macroeconomic model spanning more than four decades, this study investigates whether decentralized governance has influenced the trajectory of Kenya’s longevity transition.

2.5. Macro-Micro Linkages in Health

Finally, an important methodological gap remains in connecting macro-policy with micro-level outcomes. Classical public economics highlights that national policies interact with local behaviors (Deaton, 2015). Despite the importance of linking national trends to household outcomes, empirical studies seldom integrate long-run macroeconomic series with comprehensive census microdata. Most studies rely on DHS surveys, which lack consistent long-run macro linkages (Rutstein, 2000) or on macro analyses that overlook within-country heterogeneity (Case & Deaton, 2017).

This paper addresses that gap by constructing a dataset that links national fiscal and gender indicators to individual child survival outcomes drawn from multiple waves of Kenya’s census (IPUMS). This integrated approach makes it possible to assess how fiscal composition and gender parity jointly influence regional inequalities in longevity and the survival probabilities of children at the household level.

3. THEORETICAL FRAMEWORK

This study is grounded in a dual-layered theoretical framework that integrates a Dynamic Longevity Production Function at the macroeconomic level with a Household Production of Health Model at the microeconomic level. This approach allows for the simultaneous examination of how aggregate fiscal inputs determine national longevity trends while dissecting the heterogeneous effects of these inputs at the household level.

3.1. The Macro-Fiscal Health Production Function

To analyse the long-run determinants of life expectancy, we adapt the aggregate production function framework used by Mankiw, Romer, and Weil (1992), extended to the health sector by Fay, Leipziger, Wodon, and Yepes (2005), and further modified here to incorporate fiscal composition identities. We posit that national health status (proxied by life expectancy, LE_t) It is a slow-moving stock outcome shaped by fiscal choices, demographic pressures, and human capital accumulation over time.

Let LE_t denote life expectancy at time (t). We specify the dynamic health production function as:

$$LE_t = f(H_t^D, H_t^R, E_t, Dep_t, Z_t, \phi_{t-1}) \quad (1)$$

Where:

- H_t^D is real development (capital), health expenditure (Infrastructure, equipment).
- H_t^R is real recurrent health expenditure (Wages, medicines).
- E_t is human capital, proxied by gender parity in primary (*edgap_pri*) and secondary (*edgap_sec*) schooling.
- Dep_t is demographic pressure (Dependency ratio).
- Z_t represents institutional factors (Devolution, Free Primary Education).
- ϕ_{t-1} captures the persistence of life expectancy.

3.1.1. Fiscal Composition and the Budget Constraint

Public health spending is subject to an aggregate budget constraint.

$$G_{health,t} = H_t^D + H_t^R \quad (2)$$

Where total health expenditure $G_{health,t}$ is allocated between development and recurrent components. Crucially, we posit that it is not just the level of (G) that matters, but the fiscal composition. We define the composition ratios as:

$$dv_t = \frac{H_t^D}{G_{health,t}} \text{ (development-to-health ratio)}$$

And

$$rec_t = \frac{H_t^R}{G_{health,t}} \text{ (recurrent-to-health ratio)}$$

Since $dv_t + rec_t = 1$, they capture the fiscal composition.

Thus:

$$H_t^D = dv_t H_t, H_t^R = (1 - dv_t) H_t$$

Substituting these into the production function allows us to test the distinct elasticities of each component. The augmented logarithmic form estimated in our Vector Error-Correction Model (VECM) is:

$$LE_t = \alpha + \beta_1(H_t^D) + \beta_2(H_t^R) - \lambda E_t + \delta D_t + \gamma_r + \mu_t + v_{rt} \quad (3)$$

Where:

- β_1 and β_2 are the long-run elasticities of fiscal composition.
- λ captures the efficiency drag of gender disparity.
- γ_r are region fixed effects capturing structural differences.
- μ_t captures time-specific shocks.
- v_t is the stochastic error term (Capturing national shocks).

Theoretical Prediction 1 (Fiscal Composition): We hypothesize that $\beta_1 > \beta_2$ Standard capital theory suggests that while physical capital (H_t^D) is necessary, but it suffers from diminishing returns and depreciation. In contrast, recurrent expenditure (H_t^R) represents the "effective labor" and "consumables" required to operationalize capital. In resource-constrained settings, the marginal product of an additional nurse or drug supply (recurrent) often exceeds that of an additional hospital wing (Filmer & Pritchett, 1999).

Theoretical Prediction 2 (Demographic Crowding-Out): We incorporate a demographic constraint where high dependency ratios (Dep_t) crowd out long-run investment. Following Lee and Mason (2011), we expect $\frac{\partial H_t^D}{\partial Dep_t} < 0$, as pressure to fund immediate consumption (wages, consumables) forces a reallocation away from capital formation.

3.2. Derivation of the Micro-Level Interaction Model

The micro-level analysis is derived from the Household Production of Health theory established by Becker (1965) and formalized by Rosenzweig and Schultz (1982). Here, child survival is viewed as a commodity produced within the household using market goods and time inputs, conditional on the external environment.

Let the survival outcome (s_{ij}) of child (i) in household (j) in region (k) be produced by the following technology:

$$s_{ij} = f(M_j, G_k, X_j, \epsilon_{ij}) \quad (4)$$

Where:

- M_j is the mother's human capital (years of education).
- G_k is the public health input in the region (k) - derived from national recurrent spending.
- X_j represents other household endowments (wealth, sanitation).

- ϵ_{ij} is the child's genetic health endowment.

We are specifically interested in the interaction between the mother's education (M_j) and public inputs (G_k). The linearized estimating equation for the probability of child death ($P(\text{child_death}_{ij} = 1) = 1 - s_{ij}$) becomes:

$$\Pr(\text{child_death}_{ij} = 1 = \alpha + \phi_1 M_j + \phi_2 G_k + \phi_3 (M_j \times G_k) + X'\delta + \eta_{ij} \quad (5)$$

3.2.1. Derivation of the Substitution Effect

The coefficient of interest is the interaction term ϕ_3 , which represents the cross-partial derivative of the mortality function with respect to maternal education and public spending.

$$\frac{\partial^2 P(\text{child_death})}{\partial M \partial G} = \phi_3$$

Complementarity Hypothesis (ϕ_3): Public spending enhances the protective effect of maternal education. Educated mothers are better equipped to navigate the health system and utilize available clinics, implying inputs are complements.

Substitution Hypothesis ($\phi_3 > 0$): Public spending acts as a substitute for maternal human capital. In environments where the state fails to provide functional health systems (G_k) tend towards zero, maternal knowledge (M_j) is the sole determinant of survival (e.g., via hygiene, oral rehydration). However, as the state provides reliable medical care (i.e G_k increases) the survival advantage of being born to an educated mother diminishes because the health system protects all children regardless of maternal literacy (Glewwe, 1999).

Based on the theory of the "second best," we postulate a substitution effect ($\phi_3 > 0$), suggesting that recurrent health expenditure acts as an equalizer, reducing the survival penalty faced by children of uneducated mothers.

3.3. Mapping Theory to Empirical Strategy

This dual framework directly maps to our two-part empirical strategy.

Macro-Dynamics: The logarithmic production function (Equation 3) motivates the use of Johansen Cointegration and VECM. The VECM allows us to identify the long-run structural parameters (β_1, β_2) defined in the theory.

Micro-Interactions: The household technology derivation (Equation 5) justifies the Fixed-Effects Linear Probability Model used in the second half of the paper. By interacting the aggregate recurrent expenditure ratio with individual maternal education, we explicitly test the sign of the cross-partial derivative (ϕ_3), thereby identifying the nature of the relationship (Substitutes vs. complements) between the state and the household in health production.

4. RESULTS

The following section presents the empirical results derived from both the macroeconomic time-series analysis and the micro-level panel regressions. The findings address the critical evidence gaps regarding the impact of fiscal composition, demographic pressure, and gender disparities on longevity and child survival in Kenya.

Table 1. Long-Run VECM cointegrating equations.

Variables	Coef.	St.Err.	t-value	p-value	95% Confidence Interval	Sig.
Cointegrating equation 1 (normalized on life expectancy)						
lif_exp	1					
hlth_dv_ratio	0 (Omitted)					
hlth_rec_ratio	-28.392	12.082	2.35	0.019	[4.712, 52.073]	**
edgap_pri	-125.142	34.059	3.67	0.000	[58.388, 191.897]	***
edgap_sec	250.712	33.215	-7.55	0.000	[-315.812, -185.612]	***
dep_rat	-10.458	10.187	1.03	0.305	[-9.508, 30.424]	
cntrl_devo.	18.409	5.536	-3.33	0.001	[-29.258, -7.560]	***
cntrl_freepri	8.015	2.320	-3.45	0.001	[-12.562, -3.468]	***
_cons	68.909					
Cointegrating equation 2 (normalized on development health spending share)						
lif_exp	1.11e-16 (Omitted)					
hlth_dv_ratio	1					
hlth_rec_ratio	3.029	1.147	-2.64	0.008	[-5.278, -0.781]	***
edgap_pri	14.257	3.234	-4.41	0.000	[-20.596, -7.919]	***
edgap_sec	-24.263	3.154	7.69	0.000	[18.082, 30.444]	***
dep_rat	1.922	0.967	-1.99	0.047	[-3.818, -0.027]	**
cntrl_devo	-1.562	0.526	2.97	0.003	[0.532, 2.592]	***
cntrl_freepri	-0.460	0.220	2.09	0.037	[0.028, 0.892]	**
_cons	-2.536					

Note: Asterisks in the sig. column denote statistical significance: ** p < 0.05, *** p < 0.01.

4.1. Long-Run Macroeconomic Determinants of Longevity

Table 1 presents the cointegrating vectors from the Vector Error-Correction Model (VECM), establishing the long-run equilibrium relationships between fiscal policy, education gaps, and aggregate life expectancy.

The first long-run cointegrating Equation 1 based on Table 1 above, can be illustrated as indicated below:-

$$lif_exp_t = 68.9 - 28.4 hlth_rec_ratio_t - 125.1 edgap_pri_t + 250.7 edgap_sec_t - 10.5 dep_rat_t + 18.4 cntrl_devo_t + 8.0 cntrl_freepri_t \quad (6)$$

The primary variable of interest, the recurrent health expenditure ratio, exhibits a statistically significant and negative coefficient ($\beta = -28.39, p = 0.019$). This estimate implies that, in the long run, a reallocation of the health budget toward recurrent expenditures (wages and consumables) at the expense of development expenditures is associated with a decline in aggregate life expectancy. Conversely, this suggests that capital-intensive development spending is the primary driver of the long-term longevity frontier. This aligns with the "stock accumulation" hypothesis posited by Deaton (2015) and Jamison et al. (2013), where, while recurrent spending facilitates the immediate consumption of health services, it is the accumulation of physical health infrastructure and technology that structurally shifts survival probabilities over decades.

The control variables reveal distinct structural constraints. The gender parity gap at the secondary school level presents a large, statistically significant coefficient ($\beta = 250.71, p < 0.001$). The magnitude of this coefficient relative to the primary education gap ($\beta = -125.14$) identifies inequality in secondary education as a binding constraint on the health transition, consistent with the "missing women" framework (Klasen, 2002; Sen, 1992). The positive sign on the devolution dummy ($\beta = 18.41, p = 0.001$) indicates a positive structural break following the 2013 decentralization reforms, suggesting that the localization of health governance has shifted the intercept of the longevity function upwards (Oates, 1999).

Finally, the dependency ratio carries a negative coefficient ($\beta = -10.46$), consistent with the theoretical expectation that demographic pressure restricts fiscal space (Lee & Mason, 2011). However, this estimate is not statistically significant ($p = 0.305$) in the long-run vector, suggesting that demographic burdens may operate primarily through short-run fiscal adjustments rather than as a direct determinant of the long-run equilibrium.

Table 2. Short-Run VECM Estimates towards long-run equilibrium.

Dependent variables	Explanatory variables	Coef.	St.err.	t-value	p-value	[95% Conf Interval]	Sig.	
Δlif_exp	_ce1 L1.	0.295	0.193	1.53	0.127	-0.084	0.674	
	_ce2 L1.	2.784	1.961	1.42	0.156	-1.059	6.627	
	lif_exp LD.	0.042	0.267	0.16	0.874	-0.481	0.565	
	hlth_dv_ratio LD.	-2.416	1.716	-1.41	0.159	-5.779	0.946	
	hlth_rec_ratio LD.	3.583	1.388	2.58	0.010	0.864	6.303	***
	edgap_pri LD.	3.299	6.318	0.52	0.602	-9.085	15.682	
	edgap_sec LD.	3.09	6.401	0.48	0.629	-9.454	15.635	
	dep_rat LD.	-0.158	3.499	-0.05	0.964	-7.017	6.7	
	cntrl_devo LD.	0.347	0.721	0.48	0.631	-1.066	1.759	
	cntrl_freepri LD.	1.026	0.716	1.43	0.152	-0.379	2.43	
$\Delta hlth_dv_ratio$	_cons	0.012	0.129	0.10	0.924	-0.241	0.266	
	_ce1 L1.	-0.071	0.024	-2.92	0.003	-0.118	-0.023	***
	_ce2 L1.	-0.743	0.246	-3.02	0.002	-1.224	-0.261	***
	lif_exp LD.	0.043	0.033	1.29	0.195	-0.022	0.109	
	hlth_dv_ratio LD.	0.142	0.215	0.66	0.508	-0.279	0.563	
	hlth_rec_ratio LD.	-0.088	0.174	-0.51	0.612	-0.429	0.253	
	edgap_pri LD.	-1.716	0.791	-2.17	0.03	-3.267	-0.165	**
	edgap_sec LD.	-0.026	0.802	-0.03	0.974	-1.597	1.545	
	dep_rat LD.	1.655	0.438	3.78	0.00	0.796	2.514	***
	cntrl_devo LD.	0.034	0.09	0.38	0.704	-0.143	0.211	
$\Delta hlth_rec_ratio$	cntrl_freepri LD.	-0.069	0.09	-0.77	0.444	-0.245	0.107	
	_cons	0.014	0.016	0.88	0.377	-0.017	0.046	
	_ce1 L1.	0.011	0.027	0.42	0.676	-0.042	0.064	
	_ce2 L1.	0.151	0.274	0.55	0.581	-0.386	0.689	
	lif_exp LD.	-0.012	0.037	-0.32	0.748	-0.085	0.061	
	hlth_dv_ratio LD.	-0.033	0.24	-0.14	0.891	-0.503	0.437	
	hlth_rec_ratio LD.	-0.184	0.194	-0.95	0.342	-0.565	0.196	
	edgap_pri LD.	0.421	0.884	0.48	0.634	-1.311	2.154	
	edgap_sec LD.	-0.43	0.895	-0.48	0.631	-2.185	1.325	
	dep_rat LD.	-0.467	0.490	-0.95	0.34	-1.427	0.492	
cntrl_devo LD.	-0.023	0.101	-0.23	0.817	-0.221	0.174		
cntrl_freepri LD.	0.236	0.100	2.35	0.019	0.040	0.432	**	
_cons	-0.016	0.018	-0.89	0.374	-0.052	0.019		

Dependent variables	Explanatory variables	Coef.	St.err.	t-value	p-value	[95% Conf Interval]	Sig.	
Δ edgap_pri	_ce1 L1.	0.004	0.009	0.52	0.602	-0.012	0.021	
	_ce2 L1.	0.061	0.087	0.70	0.484	-0.109	0.230	
	lif_exp LD.	0.006	0.012	0.53	0.595	-0.017	0.029	
	hlth_dv_ratio LD.	-0.052	0.076	-0.69	0.492	-0.201	0.096	
	hlth_rec_ratio LD.	0.109	0.061	1.77	0.077	-0.012	0.229	*
	edgap_pri LD.	-0.099	0.279	-0.36	0.721	-0.646	0.447	
	edgap_sec LD.	0.085	0.283	0.30	0.764	-0.469	0.639	
	dep_rat LD.	-0.238	0.155	-1.54	0.123	-0.541	0.064	
	cntrl_devo LD.	-0.034	0.032	-1.07	0.286	-0.096	0.028	
	cntrl_freepri LD.	0.009	0.032	0.28	0.783	-0.053	0.071	
	_cons	-0.001	0.006	-0.22	0.825	-0.012	0.010	
Δ edgap_sec	_ce1 L1.	-0.001	0.005	-0.16	0.874	-0.011	0.009	
	_ce2 L1.	-0.031	0.051	-0.60	0.545	-0.130	0.069	
	lif_exp LD.	0.00	0.007	0.05	0.956	-0.013	0.014	
	hlth_dv_ratio LD.	0.029	0.044	0.66	0.506	-0.057	0.116	
	hlth_rec_ratio LD.	-0.038	0.036	-1.06	0.287	-0.108	0.032	
	edgap_pri LD.	-0.265	0.163	-1.62	0.104	-0.584	0.055	
	edgap_sec LD.	-0.068	0.165	-0.41	0.682	-0.392	0.256	
	dep_rat LD.	0.245	0.09	2.71	0.007	0.068	0.422	***
	cntrl_devo LD.	0.017	0.019	0.91	0.362	-0.020	0.053	
	cntrl_freepri LD.	-0.02	0.018	-1.06	0.29	-0.056	0.017	
	_cons	-0.008	0.003	-2.36	0.018	-0.014	-0.001	**
Δ dep_rat	_ce1 L1.	0.017	0.008	2.08	0.037	0.001	0.034	**
	_ce2 L1.	0.177	0.084	2.10	0.036	0.012	0.342	**
	lif_exp LD.	-0.006	0.011	-0.54	0.589	-0.029	0.016	
	hlth_dv_ratio LD.	-0.036	0.074	-0.49	0.622	-0.181	0.108	
	hlth_rec_ratio LD.	0.053	0.06	0.88	0.379	-0.065	0.170	
	edgap_pri LD.	0.063	0.272	0.23	0.818	-0.470	0.596	
	edgap_sec LD.	0.002	0.276	0.01	0.994	-0.538	0.542	
	dep_rat LD.	0.581	0.151	3.85	0.00	0.285	0.876	***
	cntrl_devo LD.	-0.004	0.031	-0.12	0.905	-0.065	0.057	
	cntrl_freepri LD.	-0.008	0.031	-0.24	0.807	-0.068	0.053	
	_cons	-0.003	0.006	-0.49	0.625	-0.014	0.008	
Δ cntrl_devo	_ce1 L1.	0.144	0.053	2.70	0.007	0.04	0.249	***
	_ce2 L1.	1.481	0.541	2.74	0.006	0.421	2.541	***
	lif_exp LD.	-0.129	0.074	-1.76	0.079	-0.274	0.015	*
	hlth_dv_ratio LD.	-1.1	0.473	-2.32	0.020	-2.027	-0.172	**
	hlth_rec_ratio LD.	0.632	0.383	1.65	0.099	-0.118	1.383	*
	edgap_pri LD.	1.61	1.743	0.92	0.356	-1.806	5.026	
	edgap_sec LD.	-0.108	1.766	-0.06	0.951	-3.569	3.352	
	dep_rat LD.	-0.278	0.965	-0.29	0.774	-2.17	1.614	
	cntrl_devo LD.	-0.01	0.199	-0.05	0.958	-0.400	0.379	
	cntrl_freepri LD.	0.214	0.198	1.08	0.279	-0.174	0.601	
	_cons	-0.004	0.036	-0.10	0.919	-0.074	0.066	
Δ cntrl_freepri	_ce1 L1.	0.078	0.05	1.55	0.12	-0.02	0.176	
	_ce2 L1.	0.645	0.508	1.27	0.204	-0.351	1.641	
	lif_exp LD.	-0.076	0.069	-1.10	0.27	-0.212	0.059	
	hlth_dv_ratio LD.	-0.210	0.445	-0.47	0.636	-1.082	0.661	
	hlth_rec_ratio LD.	-0.286	0.360	-0.80	0.426	-0.991	0.419	
	edgap_pri LD.	-3.509	1.637	-2.14	0.032	-6.717	-0.300	**
	edgap_sec LD.	-1.052	1.658	-0.63	0.526	-4.303	2.198	
	dep_rat LD.	0.61	0.907	0.67	0.501	-1.167	2.387	
	cntrl_devo LD.	0.213	0.187	1.14	0.255	-0.153	0.579	
	cntrl_freepri LD.	0.091	0.186	0.49	0.622	-0.272	0.455	
	_cons	-0.024	0.034	-0.72	0.473	-0.09	0.042	
	Mean dependent var		-0.001	SD dependent var		0.027		
	Number of obs .		37.000	Akaike crit. (AIC)				

Note: Asterisks in the Sig. column denote statistical significance: * p < 0.10, ** p < 0.05, *** p < 0.01.

4.2. Short-Run Adjustment Dynamics

The error-correction terms (Δlif_exp equations) in Table 2 suggest a slow adjustment process for life expectancy, which is consistent with the biological lag inherent in health outcomes. A distinct asymmetry characterizes the system's dynamics. The fiscal variables adjust rapidly to deviations from equilibrium, with the recurrent health ratio correcting approximately 74% of the disequilibrium within a single period. In contrast, the error-correction term for life expectancy is statistically insignificant. This result supports the characterization of longevity as a slow-moving "stock variable" (Bleakley, 2010); while fiscal policy is highly elastic to shocks, health outcomes exhibit significant inertia and do not adjust instantaneously to annual budget fluctuations.

Table 3. Macro-Micro Region Fixed-Effects Regressions.

Variables	(1)	(2)	(3)
	Share65	Mean_child_mort	Mean_age
hlth_rec_ratio	0.112*** (0.022)	0.031*** (0.004)	25.618*** (6.966)
dep_rat	0.013*** (0.002)	-0.026*** (0.003)	3.644*** (0.385)
edgap_pri	-0.397*** (0.077)	0.000 (.)	-94.578*** (26.071)
edgap_sec	0.000 (.)	0.000 (.)	0.000 (.)
_cons	-0.046*** (0.015)	0.044*** (0.005)	2.791 (4.339)
N	11044974	7203039	11044974

Note: Asterisks in the Sig. column denotes statistical significance: *** $p < 0.01$.

4.3. Macro-Micro Linkages: Regional Longevity and Child Survival

The analysis in Table 3 shows that the Recurrent Health Expenditure Ratio significantly predicts the Share of the Elderly (65+) population ($\beta = 0.112, p < 0.001$). This confirms that regions exposed to periods of higher national recurrent spending see a robust preservation of their elderly cohorts, likely due to better management of chronic, age-related conditions that require continuous care rather than one-off interventions (Bloom & Canning, 2000).

Interestingly, the association with Mean Child Mortality is positive ($\beta = 0.031, p < 0.001$). While counterintuitive, this may reflect survival selection effects or the concurrent expansion of reporting systems in high-spending periods. Alternatively, it highlights a divergence where fiscal policy protecting the elderly does not automatically translate to identical gains in neonatal survival without targeted maternal interventions (Cutler et al., 2006).

Table 4. Interaction between maternal education and recurrent health expenditure.

Variables	(1)	(2)	(3)
	Baseline	+ Region FE	+ Macro controls
Mother's education (Years)	-0.002 (0.002)	-0.003 (0.002)	-0.006*** (0.001)
Interaction: Edu x recurrent health Exp	0.001 (0.004)	0.002 (0.004)	0.007** (0.003)
Urban household	-0.071*** (0.014)	-0.057*** (0.009)	-0.030*** (0.009)
Dependency ratio of non-working to working			-0.389*** (0.038)
Gender parity at the secondary level			4.142*** (0.128)
Observations	1872997	1872997	1872997
R-squared	0.073	0.111	0.154

Note: Asterisks in the Sig. column denote statistical significance: ** $p < 0.05$, *** $p < 0.01$.

4.4. Micro-Level Interactions: Maternal Education and Fiscal Context

Consistent with human capital theory, maternal education is negatively associated with child mortality ($\beta = -0.006, p < 0.001$). However, the interaction between maternal education and the national recurrent health expenditure ratio is positive and significant ($\beta = 0.007, p < 0.05$). This positive interaction term indicates a substitution effect: as the state's investment in recurrent health inputs increases, the marginal protective return to maternal education diminishes. This suggests that public health spending effectively substitutes for household human capital in producing child survival, acting as an alternative safety net for children of uneducated mothers (Glewwe, 1999; Schultz, 1999).

Furthermore, the dependency ratio (non-working to working population) appears with a protective negative coefficient ($\beta = -0.389, p < 0.001$). In the African context, this challenges the standard "dependency burden"

narrative, suggesting that larger extended family structures (which often drive higher dependency ratios) may provide essential childcare support and risk pooling that improves child survival rates (Maitra, 2004).

4.5. Further Micro-Heterogeneity: The "Urban Advantage" and Structural Constraints

Beyond the interaction effects, the micro-level estimations reveal two critical structural magnitudes that contextualize the impact of fiscal policy.

4.5.1. The "Urban Equivalent" of Maternal Education

The results in Table 4 also allow us to quantify the "urban advantage" in terms of human capital. The coefficient for the Urban Household dummy is negative and highly significant ($\beta = -0.030, p < 0.001$), indicating that, holding all else constant, residing in an urban area reduces the probability of child death by 3.0 percentage points. Comparing this to the Maternal Education coefficient ($\beta = -0.006$), we observe a striking equivalence where residing in an urban centre confers a survival advantage equivalent to approximately 5 additional years of maternal schooling ($\frac{0.030}{0.006} = 5$).

This finding suggests that urban infrastructure (proximity to hospitals, sanitation, and information) acts as a massive substitute for individual human capital. For policymakers, this implies that in rural areas (where this locational dividend is absent), the fiscal injection of recurrent health expenditure is structurally urgent to bridge this 5-year human capital gap.

4.5.2 The Lethality of the Secondary Gender Gap

While the VECM analysis identified the gender gap as a long-run drag on longevity, the micro-results quantify its immediate lethality. The coefficient for the Gender Parity Gap at Secondary Level in the child mortality regression is exceptionally large and positive ($\beta = 4.142, p < 0.001$). This indicates that children born in regions with high gender disparity in secondary schooling face a disproportionately high risk of mortality, independent of their own mother's education level.

This points to a "community-level externality" of gender inequality. Even if an individual mother is educated, child survival in a region where women generally lack secondary education (a high gap) is generally low. This supports the "diffusion of innovations" theory, where female secondary education creates a community-wide knowledge spillover that protects all children, not just those of the educated elite.

4.5.3. The Role of Macro-Fiscal and Demographic Environment

Finally, the progression from Model 1 (Baseline) to Model 3 (Full Macro Controls) in Table 4 sees the R-squared triple from 0.050 to 0.154. This suggests that two-thirds of the explainable variation in child survival in our model is driven not by household characteristics alone, but by their interaction with the macro-fiscal and demographic environment. The results provide strong statistical support for the study's central premise, showing that individual survival outcomes are fundamentally influenced by the macro-level policy.

4.6. Robustness and Sensitivity Analyses

We conducted robustness checks, and the sensitivity of the results is robust to alternative education definitions as well as the exclusion of individual census waves, as presented in Tables A11 and A12 in the Appendix 1 section. The results from the tables indicate that the key coefficients retain sign, magnitude, and significance, indicating that the findings are stable and are not driven by any single period.

5. DISCUSSION

A natural concern when studying health outcomes is whether the results simply restate the well-known association between development, individual characteristics, and survival. The evidence presented here goes beyond such correlations by isolating which components of fiscal policy matter, over what horizon, and for whom. The macroeconomic results show that long-run longevity responds to capital accumulation rather than to operational spending, while the micro-level evidence reveals that recurrent expenditure reshapes the distribution of survival by substituting for maternal education. These findings imply that aggregate correlations between spending and health conceal opposing mechanisms operating at different time scales and levels of aggregation. Ignoring this structure risks misleading policy prescriptions, particularly in African settings characterized by fiscal rigidity and deep household heterogeneity.

This study provides a comprehensive empirical analysis of the fiscal, demographic, and gender-based determinants of longevity and child survival in Kenya. By leveraging both macroeconomic time-series data (1981-2023) and micro-level census data, the findings bridge critical gaps in the literature regarding how national policy choices interact with household characteristics to shape health outcomes. The discussion below interprets the results in the context of existing theoretical and empirical work.

5.1. The Fiscal Composition Puzzle: Capital Accumulation Versus Consumption

A central contribution of this analysis is the provision of rigorous evidence on how the composition of health expenditure influences longevity. The Vector Error-Correction Model (VECM) reveals a robust long-run relationship between fiscal composition and life expectancy. The recurrent health expenditure ratio carries a significant negative coefficient ($\beta = -28.39, p = 0.019$). This indicates that, over the long run, a fiscal shift toward recurrent spending on wages and consumables, at the expense of development expenditure, is detrimental to aggregate life expectancy. In

contrast, capital accumulation through investments in hospitals, sanitation systems, and medical technology emerges as the primary driver of the longevity transition.

This finding challenges the liquidity argument often favoured in short-term policy debates. While recurrent spending is necessary to sustain daily operations, the results suggest that it functions more as health consumption, whereas development spending functions as health investment. This interpretation aligns with the stock accumulation theory of health advanced by Deaton (2015) and Jamison et al. (2013). Their work emphasizes that sustained improvements in mortality are driven by the gradual accumulation of a health capital stock, such as infrastructure and technology, rather than by temporary fluctuations in operational spending. When governments prioritize wage bills over infrastructure expansion, a common outcome in political economy settings, they effectively consume resources that could otherwise advance the epidemiological frontier.

The short-run dynamics add further nuance to this structural view. The VECM error-correction terms show that fiscal aggregates adjust rapidly to shocks, with an estimated adjustment speed of 74 percent per year. Life expectancy, however, exhibits no statistically significant short-run adjustment. This supports the characterization of longevity as a slow-moving stock variable, consistent with Bleakley (2010). Survival outcomes evolve gradually through cohort replacement and sustained exposure to improved infrastructure, whereas fiscal policy remains highly elastic. The implication is that recurrent spending may satisfy immediate political demands, but it is the more durable investment in physical capital that secures long-term survival gains.

5.2. Gender Parity as a Macro-Determinant of Longevity

The results strongly support the "missing women" and human capital theories (Sen, 1992). The gender parity gap at the secondary school level is identified as a severe structural drag on aggregate life expectancy ($\beta = 250.71$). This magnitude suggests that gender inequality is not merely a social justice issue but a binding macroeconomic constraint. This finding resonates with Klasen (2002), who demonstrated that gender inequality in education directly lowers economic growth and health outcomes by suppressing the "demographic dividend." When women are denied secondary education, their agency in health decision-making is curtailed, leading to higher fertility rates and poorer child health outcomes, which in turn depresses aggregate life expectancy. The contrast with the primary education gap suggests a threshold effect: basic literacy may be necessary but is insufficient for modern health transitions; it is secondary education that confers the critical thinking required to navigate complex health systems, consistent with the "maternal education channel" described by Caldwell (1979).

5.3. The Substitution Effect: Fiscal Policy as a Safety Net

Perhaps the most novel contribution of this study is the micro-level evidence of a substitution effect between maternal education and public health spending. The interaction term in the child mortality model is positive and significant ($\beta = 0.007, p = 0.04$). This implies that while maternal education reduces child mortality, its marginal benefit diminishes as government recurrent health expenditure increases.

This finding nuances the standard "complementarity" hypothesis (Cutler & Lleras-Muney, 2010), which posits that educated mothers are better at using health inputs. Instead, our results suggest that robust public health systems can substitute for low maternal human capital. In regions where the state provides functional clinics, the survival of a child depends less on the mother's individual knowledge and more on the systemic quality of care. Conversely, in marginalized areas where the state is absent, the mother's education becomes the sole line of defense. This is consistent with a "development-transition" perspective: early in the transition, education compensates for weak health environments; later, as systems improve, the survival gap between educated and uneducated mothers narrows (Glewwe, 1999; Schultz, 1999).

5.4. Demographic Pressure and the "Extended Family" Dividend

The negative coefficient for the dependency ratio in the child mortality regression ($\beta = -0.389$) offers a vital counter-narrative to the "dependency burden" hypothesis prevalent in Western economic models (Bloom & Canning, 2000). In the Kenyan context, a higher dependency ratio appears protective for child survival.

This likely captures the "kinship support" mechanism described by Maitra (2004), where extended family members provide childcare, nutritional buffering, and risk pooling that protect vulnerable children. Far from being a mere drain on resources, the non-working population in African households contributes essential non-market labor (caregiving). However, at the macro-level, the negative (though insignificant) sign of demographic pressure in the VECM aligns with research emphasizing that high dependency burdens eventually constrain the fiscal space required for the long-horizon capital investments identified in Section 5.1 (Lee & Mason, 2011).

5.5. Devolution and Structural Breaks

Finally, the significance of the devolution dummy ($\beta = 18.41$) confirms that the 2013 decentralization of health services in Kenya marked a positive structural break in longevity trends. While implementation challenges exist, the localization of decision-making appears to have successfully disrupted prior stagnation, potentially by aligning health spending more closely with local disease burdens. This supports theoretical arguments for fiscal federalism in health (Oates, 1999), suggesting that moving decision rights closer to the user yields long-term welfare gains despite initial administrative friction.

5.6. Conclusion

In summary, these results suggest that the path to longevity in Kenya requires a strategic rebalancing. While micro-level evidence shows that recurrent spending acts as an essential equity mechanism (substituting for maternal education), the macro-level evidence warns that over-reliance on consumption spending at the expense of capital accumulation stagnates the long-run frontier. Sustainable health improvement, therefore, demands a "twin-track" approach: ring-fencing capital budgets to build the "health stock" for the future, while targeting recurrent subsidies specifically toward marginalized regions to bridge the human capital gap today (Bloom et al., 2011; Jamison et al., 2013).

6. POLICY IMPLICATIONS

The empirical results of this study highlight a complex policy landscape shaped by a clear intertemporal trade-off. Macroeconomic evidence indicates that long-run improvements in longevity are driven primarily by capital accumulation through development expenditure. In contrast, microeconomic evidence shows that operational liquidity in the form of recurrent expenditure functions as a critical safety net for vulnerable households. This duality suggests that the prevailing development paradigm, which often oscillates between infrastructure expansion and wage-bill growth, requires a more calibrated twin-track approach.

6.1. Correcting the Fiscal Bias: Ring-Fencing Capital Accumulation

The VECM analysis identifies a significant negative long-run association between the recurrent expenditure ratio and life expectancy ($\beta = -28.39$, $p = 0.019$). This finding implies that capital expenditure is the true driver of the longevity transition. It challenges the political economy tendency in Kenya to prioritize short-term recurrent consumption over long-term investment.

The National Treasury and County Treasuries should institutionalize fiscal rules that prevent the crowding out of capital projects. The Medium-Term Expenditure Framework (MTEF) should enforce a capital floor, mandating that a fixed share of health allocations remain dedicated to infrastructure and technology acquisition. Without sustained investment in diagnostic equipment, specialized units, and sanitation systems, the health system will stagnate regardless of staffing levels (Jamison et al., 2013).

6.2. Leveraging Recurrent Spending as an Equity Mechanism

While capital investment drives the national frontier, the micro-level findings reveal that recurrent spending is the primary protector of marginalized households. The significant substitution effect ($\beta = 0.007$, $p < 0.05$) shows that in regions with low maternal literacy, government operational spending effectively substitutes for household human capital in producing child survival.

Health financing should adopt a formula-based deprivation index for recurrent allocations. Rather than blanket increases in recurrent spending, operational funds should be disproportionately targeted toward counties with high maternal illiteracy. Concentrating resources in these areas through staff and commodities can break the link between maternal deprivation and child mortality without undermining the national capital budget (Glewwe, 1999).

6.3. Redefining Female Secondary Education as a Public Health Priority

The study identifies female secondary education, measured here as parity, as a significant driver of life expectancy, with a large positive coefficient ($\beta = 250.71$, $p < 0.001$). This result points to a threshold effect. While primary education was sufficient to reduce basic mortality in earlier decades, the gains from basic literacy have largely been exhausted. The longevity frontier now depends on the agency and cognitive skills that are fostered during adolescence. Primary education provides functional literacy, such as the ability to read a label, but secondary education develops critical thinking and executive function. These advanced cognitive skills are essential for navigating the modern epidemiological transition. Tasks such as managing chronic conditions like diabetes and hypertension, adhering to antiretroviral therapies, and exercising autonomous reproductive choices within the household require higher levels of cognitive capacity. As the burden of disease shifts from simple infections to complex lifestyle factors, the health production function becomes more cognitively demanding. Female secondary retention, therefore, emerges as a non-negotiable input for future survival gains.

The Ministries of Health and Education should coordinate to treat female secondary retention as a high-return health intervention. Policy must move beyond enrollment targets and focus on transition and retention incentives. Conditional Cash Transfers for girls' secondary completion and the removal of hidden costs, such as uniforms and examination fees, are critical tools. Given the magnitude of the coefficient, the return on investment in female education likely exceeds that of marginal medical inputs, supporting the concept of education as a social vaccine in health policy (Lutz & KC, 2011).

6.4. Harnessing the Kinship Dividend while Managing Fiscal Pressure

The findings reveal a paradox. High dependency ratios are protective at the household level through kinship care ($\beta = -0.389$, $p < 0.05$) but simultaneously strain fiscal space at the macro level. Social protection policies should formalize the kinship economy to relieve fiscal pressure. Scaling up programs such as Inua Jamii (cash transfers for the elderly) can subsidize childcare provided by extended families, enhancing the protective effect at the micro level. At the same time, the state must broaden the tax base beyond labor income to mitigate the fiscal burden imposed by a large

dependent population, ensuring that demographic weight does not erode public investment capacity (Bloom et al., 2011).

6.5. Deepening Fiscal Federalism

The structural break associated with the 2013 devolution reform ($\beta = 18.41$, $p = 0.001$) confirms that decentralization has generated welfare gains. Sustaining these gains requires that local governments avoid replicating the national bias toward recurrent consumption.

The Commission on Revenue Allocation (CRA) should strengthen the conditionality of health grants. Future disbursements should be linked to performance-based indicators that reward counties for maintaining an optimal balance between capital health expenditure and recurrent health expenditure (operational) execution. This would incentivize local governments to act as responsible stewards of the long-term health stock rather than focusing narrowly on payroll expansion (Oates, 1999).

7. LIMITATIONS AND AREAS FOR FUTURE RESEARCH

7.1. Limitations of the Study

While this study employs a novel dual-layered empirical strategy, a few limitations must be acknowledged to properly contextualize the findings.

First, the study relies on National Treasury data, which historically omits significant off-budget donor financing. In Kenya, as in many Sub-Saharan economies, a substantial share of health capital funding is provided by external partners, including USAID and The Global Fund, and often flows directly to NGOs or parallel supply chains (Lu et al., 2010). If donor resources are systematically directed toward specific capital projects, the estimates of fiscal elasticity may partially conflate domestic fiscal effort with external aid volatility.

Finally, while the micro-level region-fixed effects model controls for time-invariant unobservables such as geography and culture, it cannot fully account for time-varying regional confounders. For example, regions with higher maternal education may also have received preferential infrastructure investments linked to political patronage during particular election cycles. In such cases, the estimated substitution effect could be partially endogenous.

7.2. Areas for Future Research

The findings of this study, particularly the tension between capital accumulation and recurrent liquidity, open several promising avenues for future research in government and economics.

One important area concerns the fungibility of donor aid. Research should explicitly model the interaction between donor health funding and domestic fiscal composition. Examining whether donor support for development spending leads governments to reallocate their own resources toward recurrent consumption, or whether it stimulates additional domestic investment, would clarify the extent of fiscal displacement and refine recommendations for international partners.

A second avenue involves climate shocks and fiscal resilience. Given the sensitivity of child survival to drought and food insecurity in the Horn of Africa, and the protective role of extended family structures identified in this study, future models should incorporate climate variables. Understanding how fiscal composition mitigates or amplifies the mortality effects of climate shocks would make a valuable contribution to the emerging climate-health literature.

Third, future research should explore the interaction between public and private health provision. Kenya has a robust private and faith-based health sector, yet little is known about how public fiscal policy shapes private utilization. Investigating whether high levels of public capital spending encourage greater reliance on private services by improving trust, or whether the public sector substitutes for private care, would deepen understanding of the complementarities and trade-offs between these systems.

Finally, while this study controls for the mother's current socioeconomic status, it does not account for the intergenerational transmission of health shocks. Future research should examine how maternal early-life disease exposure and economic conditions shape current child survival outcomes. Building on the Fetal Origins Hypothesis (Almond & Currie, 2011; Barker, 1990), it's plausible that mothers born during periods of drought or economic recession, such as the 1984 famine or the structural adjustment era of the 1990s, carry latent biological scars. These may include reduced stature or metabolic compromise that influence the survival chances of their children, independent of maternal education. Analysing cohort-specific environmental exposures would provide valuable insights into how policy failures in one generation reverberate into the mortality rates of the next. Such work would deepen understanding of the long-term consequences of macroeconomic shocks and strengthen the case for policies that protect early-life health as a foundation for intergenerational human capital accumulation.

8. CONCLUSION

This study set out to examine the fiscal, demographic, and gender-based determinants of longevity in Kenya, with the aim of clarifying how public resources should be allocated to maximize health outcomes. By combining a macro-level Vector Error-Correction Model (VECM) with micro-level region-fixed effects analysis, the findings reveal a clear intertemporal trade-off that defines the political economy of health in developing countries.

At the macroeconomic level, the evidence challenges the short-term bias that often shapes fiscal planning. The significant negative long-run association between the recurrent expenditure ratio and life expectancy indicates that the longevity transition is driven primarily by capital accumulation. This implies that investments in infrastructure, technology, and sanitation systems provide the foundation for sustained improvements in survival. A fiscal strategy

that leans heavily toward consumption, such as wages and operations, at the expense of development investment, may satisfy immediate demands but fails to advance the structural epidemiological frontier. National health status is therefore best understood not as a flow of daily services but as an accumulated stock variable that requires sustained, multi-decade investment.

The microeconomic evidence provides an important counterbalance. Recurrent expenditure plays a critical equity role, functioning as a safety net that substitutes for household human capital in producing child survival. The identification of a significant substitution effect shows that operational public health spending can offset the disadvantages faced by children in households where maternal education is low. In such contexts, the presence of the state is often decisive for survival. The analysis also highlights the secondary education gender gap as a binding constraint, with community-level inequality exerting a severe effect on child survival comparable to household-level deprivation.

Taken together, these findings suggest that sustainable health improvements in Sub-Saharan Africa require a twin-track fiscal strategy. Governments should institutionalize fiscal rules that protect capital budgets in order to secure the long-term health stock, while at the same time directing recurrent liquidity toward marginalized regions to bridge immediate human capital gaps. For policymakers, the lesson is clear: operational spending saves lives in the present, but only capital investment and female secondary education can secure the longevity of future generations. Progress depends on shifting the health financing paradigm from short-term consumption toward long-term structural investment.

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Transparency: The authors state that the manuscript is honest, truthful, and transparent, that no key aspects of the investigation have been omitted, and that any differences from the study as planned have been clarified. This study followed all writing ethics.

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Appendix 1.

Table A1. Summary statistics of macro-fiscal, education, and demographic variables.

Variables	Obs.	Mean	Sd.	Min.	Max.	Skewness	Kurtosis
lif_exp	43	59.543	2.489	55.478	63.646	-0.209	1.819
hlth_dv_ratio	43	0.268	0.139	0.049	0.577	0.605	2.384
hlth_rec_ratio	43	0.770	0.102	0.517	0.951	-0.701	3.065
edgap_pri	43	0.051	0.044	-0.010	0.134	0.573	2.136
edgap_sec	39	0.095	0.060	-0.003	0.255	0.703	3.103
dep_rat	43	1.453	0.187	1.211	1.830	0.382	2.235

We begin by describing the macroeconomic and demographic environment underpinning the analysis. Table A1 presents summary statistics for life expectancy, health expenditure composition, gender gaps in education, and the dependency ratio over the study period.

Life expectancy averages approximately 59.5 years, with relatively modest variation, reflecting gradual but sustained improvements in survival over time. Health spending in Kenya is heavily skewed toward recurrent expenditures, which account for nearly 77 percent of total health spending on average, compared to 27 percent allocated to development expenditure. This imbalance highlights the policy relevance of expenditure composition rather than aggregate spending alone.

Gender disparities in education are substantially larger at the secondary level than at the primary level, underscoring the importance of distinguishing between foundational and post-foundational human capital investments. The dependency ratio remains persistently high, reflecting Kenya's youthful population structure and ongoing demographic transition.

Overall, the distributions exhibit no extreme skewness or kurtosis, supporting the suitability of the data for dynamic time-series and panel estimation.

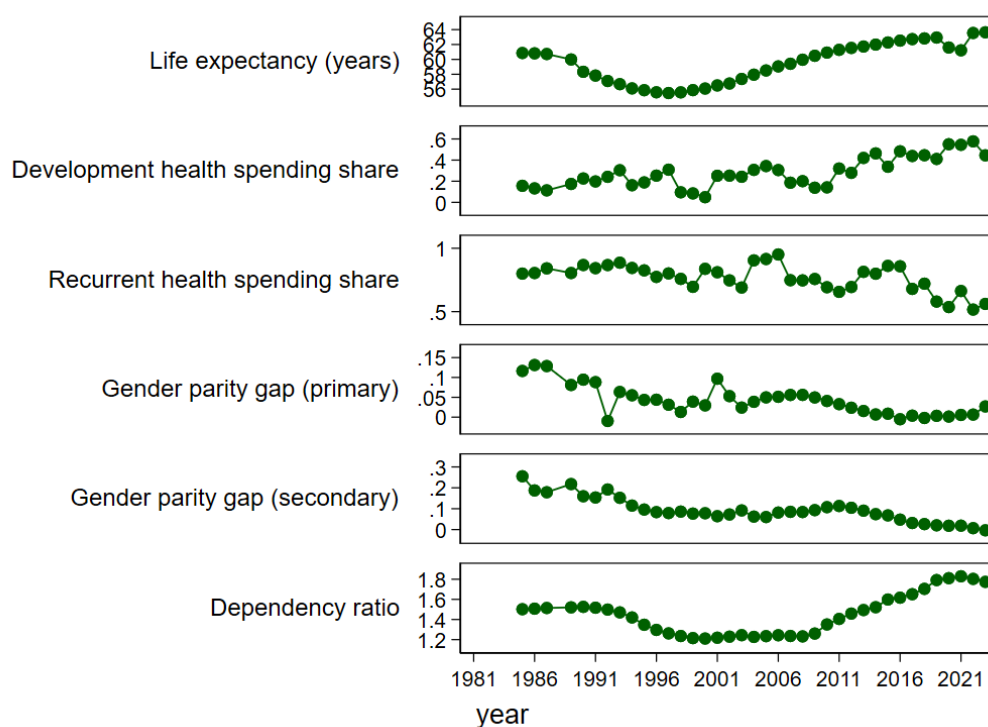


Figure A1. Trends in life expectancy, health expenditure composition, education gender gaps, and dependency ratio.

Figure A1 illustrates pronounced structural changes over time. Life expectancy declined during the late 1980s and 1990s, which coincided with macroeconomic contraction and the HIV/AIDS epidemic, before rising steadily from the early 2000s onward. Over the same period, development health expenditure gradually increased while recurrent expenditure declined, particularly after the mid-2000s.

Both primary and secondary education gender gaps narrowed substantially, consistent with education reforms and targeted gender-equity policies. These concurrent shifts motivate a dynamic framework that jointly models health outcomes, fiscal composition, education, and demographic pressures.

Table A2. Pairwise correlations among key macro variables.

Variables	(1)	(2)	(3)	(4)	(5)	(6)
(1) lif_exp	1.000					
(2) hlth_dv_ratio	0.548*	1.000				
	(0.000)					
(3) hlth_rec_ratio	-0.453*	-0.414*	1.000			
	(0.002)	(0.006)				
(4) edgap_pri	-0.152	-0.643*	0.342*	1.000		
	(0.329)	(0.000)	(0.025)			
(5) edgap_sec	-0.255	-0.646*	0.520*	0.703*	1.000	
	(0.118)	(0.000)	(0.001)	(0.000)		
(6) dep_rat	0.733*	0.627*	-0.472*	-0.186	-0.205	1.000
	(0.000)	(0.000)	(0.001)	(0.231)	(0.211)	

Note: Asterisks in the Sig. column denotes statistical significance: * $p < 0.10$.

Table A2 indicates that life expectancy exhibits a strong and positive correlation with the dependency ratio (0.733, $p < 0.01$) and development health expenditure ratio (0.548, $p < 0.01$), suggesting that higher development-oriented health spending and lower dependency burdens are associated with improved population longevity. Conversely, life expectancy is negatively correlated with the recurrent health expenditure ratio (-0.453, $p < 0.01$), which is consistent with the notion that recurrent-heavy health budgets, typically dominated by wages, administration, and consumables, may have limited long-run effects on mortality reduction. The education gender-gap variables show moderate but statistically significant correlations with health expenditure patterns, with larger gender gaps associated with higher recurrent spending and lower development spending. These relationships align with theoretical expectations that gender disparities in schooling often co-occur with weaker investment in human capital, enhancing public services. Additionally, the intercorrelations among the explanatory variables do not suggest problematic linear dependence, but they align closely with the theoretical framework motivating the subsequent cointegration analysis. Although several correlations are significant, none approach unity, indicating that multicollinearity is unlikely to distort parameter estimates in the VECM framework.

Table A3. Lag selection criteria.

Lag	LL	LR	df	p	FPE	AIC	HQIC	SBIC
0	163.901				7.9e-12	-8.535	-8.443	-8.274
1	361.599	395.39	36	0.000	1.3e-15	-17.276	-16.631*	-15.447*
2	401.301	79.405*	36	0.000	1.3e-15*	-17.476*	-16.279	-14.080

Note: * denotes the optimal lag length in the criteria.

Table A3 reports the lag selection diagnostics used to determine the appropriate lag structure for the vector autoregressive representation underlying the VECM. The information criteria indicate that the optimal lag length is the one marked with an asterisk in the table. At this lag, the Akaike Information Criterion reaches its minimum value while the Schwarz Bayesian Information Criterion and the Hannan Quinn Criterion provide consistent support for the same specification. The selection of this lag length ensures that the dynamic structure of the model adequately captures the temporal relationships among the variables without introducing unnecessary parameters. This lag choice forms the basis for the subsequent Johansen cointegration tests and the estimation of the VECM system.

Table A4. Johansen Cointegration Tests.

Max. rank	Parms	LL	Eigenvalue	Trace Statistic	5% Level	1% Level	Max Eigenvalue	5% Critical value	1% Critical value
0	42	388.334	-	125.934	94.15	103.18	48.699	39.37	45.10
1	53	362.683	0.732	77.236	68.52	76.07	29.829	33.46	38.77
2	62	377.597	0.553	47.407* ¹	47.21	54.46	25.074	27.07	32.24
3	69	390.134	0.491	22.333* ⁵	29.68	35.65	12.455	20.97	25.52
4	74	396.361	0.286	9.879	15.41	20.04	9.794	14.07	18.63
5	77	401.258	0.233	0.085	3.76	6.65	0.085	3.76	6.65
6	78	401.301	0.002	-	-	-	-	-	-

Note: Asterisks in the Sig. column denotes statistical significance: * $p < 0.10$.

Table A4 presents the results of the Johansen cointegration tests used to assess whether a stable long-run relationship exists among the variables included in the model. The trace statistic indicates the presence of cointegration at rank two at the one percent significance level, as denoted by the notation in the table. This result implies that the null hypothesis of no cointegration is rejected at conventional significance levels. The presence of two cointegrating vectors confirms that the variables share long-run equilibrium relationships despite short-run fluctuations. This finding justifies the use of the Vector Error Correction Model framework, which allows the analysis to capture both the long-run equilibrium relationships and the short-run adjustment dynamics among the fiscal and demographic variables.

Table A5. Normality test.

Skewness and Kurtosis tests for normality					
Variable	Obs	Pr(skewness)	Pr(kurtosis)	----- joint test -----	
				Adj chi2(2)	Prob > chi2
r	39	0.082	0.313	4.200	0.122
Shapiro–Wilk W test for normal data					
Variable	Obs	W	V	z	Prob>z
r	39	0.968	1.248	0.465	0.321

Table A5 indicates the results of the residual normality diagnostics using both the Skewness–Kurtosis test and the Shapiro–Wilk test. The Skewness–Kurtosis test shows a p-value of 0.082 for skewness and 0.313 for kurtosis, with a joint adjusted chi-square statistic of 4.200 ($p = 0.122$). Because all p-values exceed the conventional 5% significance threshold, we fail to reject the null hypothesis that the residuals are normally distributed. Similarly, the Shapiro–Wilk test reports a W-statistic of 0.968 with an associated p-value of 0.321, which again indicates no statistically significant departure from normality. These results confirm that the VECM residuals conform adequately to the assumption of normality, supporting the validity of subsequent inference based on the estimated model.

Table A6. Census data summary statistics.

Variables	Mean	SD	Min.	Max.	Obs.
1989					
Share of 65+ in population (Weighted)	0.03	0.01	0.00	0.05	192334
Mean child mortality rate (At mother-level)	0.06	0.03	0.00	0.13	192334
Mean age	21.58	1.59	16.80	33.78	192334
Total children ever born (f+m)	5.20	7.21	1.00	128.00	153474
Completed secondary or more	0.99	0.10	0.00	1.00	192334
1999					
Share of 65+ in population (Weighted)	0.03	0.01	0.00	0.06	239093
Mean child mortality rate (at the mother-level)	0.07	0.03	0.00	0.17	239093
Mean age	21.79	1.54	18.99	25.71	239093
Total children ever born (f+m)	4.04	2.83	1.00	18.00	239093
Completed secondary or more	1.00	0.00	1.00	1.00	239093
2009					
Share of 65+ in population (Weighted)	0.04	0.02	0.01	0.20	638939
Mean child mortality rate (At mother-level) ¹	-	-	-	-	-
Mean age	22.41	2.15	18.00	37.90	638939
Total children ever born (f+m)	3.74	2.54	1.00	16.00	638939
Completed secondary or more	1.00	0.00	1.00	1.00	638939
2019					
Share of 65+ in population (Weighted)	0.04	0.02	0.01	0.11	802631
Mean child mortality rate (At mother-level)	0.03	0.02	0.00	0.09	802631
Mean age	24.24	2.79	15.64	32.10	802631
Total children ever born (f+m)	3.14	2.08	1.00	16.00	802631
Completed secondary or more	0.99	0.09	0.00	1.00	802631
Total					
Share of 65+ in population (Weighted)	0.04	0.02	0.00	0.20	1872997
Mean child mortality rate (At mother-level)	0.04	0.03	0.00	0.17	1234058
Mean age	23.03	2.58	15.64	37.90	1872997
Total children ever born (f+m)	3.64	3.14	1.00	128.00	1834137
Completed secondary or more	1.00	0.07	0.00	1.00	1872997

Table A6 summarizes the key micro-level variables from the Kenyan census data used in the macro-micro analysis. The sample includes women of reproductive age (15–49 years) across the 1989, 1999, 2009, and 2019 waves, yielding about 1.87 million observations. The share of the population aged 65 and above rises from roughly 3 percent in 1989 and 1999 to about 4 percent in 2009 and 2019, with later waves showing greater regional variation. Child mortality at the mother level declines from 6–7 percent in 1989 and 1999 to about 3 percent in 2019, although data for 2009 are unavailable. Average age increases from 21.6 years in 1989 to over 24 years in 2019, while fertility falls from 5.2 to 3.1 children ever born. Educational attainment is high across all waves, with near-universal completion of secondary education or higher. These statistics capture major demographic, fertility, and survival transitions, providing the empirical foundation for linking individual outcomes to evolving macroeconomic and fiscal conditions.

¹ Note that region-level child mortality estimates are unavailable for the 2009 census due to missing fertility and survival data. Regressions using child mortality exclude 2009, while analyses of other outcomes use all census waves. This restriction ensures consistency without imposing assumptions on mortality trends.

Table A7. Interaction between maternal education and development health expenditure.

Variables	(1)	(2)	(3)
	Baseline	+ Region FE	+ Macro Controls
Mother's education (Years)	-0.003*** (0.001)	-0.003*** (0.001)	-0.002** (0.001)
Interaction: Edu x development health exp	0.007*** (0.002)	0.007*** (0.002)	0.005*** (0.002)
Urban household	-0.078*** (0.014)	-0.065*** (0.009)	-0.030*** (0.009)
Dependency ratio of non-working to working			1.501*** (0.055)
Gender parity at the secondary level			0.150** (0.066)
Observations	1872997	1872997	1872997
R-squared	0.050	0.088	0.154

Note: Asterisks in the Sig. column denote statistical significance: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A7 reports the robustness of the estimated relationship between maternal education and development health expenditure across three model specifications. Column (1) presents the baseline specification without fixed effects, column (2) introduces regional fixed effects, and column (3) further includes macro-level controls. The results show that the main findings remain stable across these alternative specifications.

In the baseline model, maternal education is negatively associated with child mortality, with a coefficient of ($-0.003, p < 0.01$). When regional fixed effects are introduced in column (2), the coefficient remains unchanged at ($-0.003, p < 0.01$). In the full specification with regional fixed effects and macro controls, the coefficient becomes ($-0.002, p < 0.05$). This consistency indicates that the negative association between maternal education and child mortality is robust to the inclusion of regional heterogeneity and macro-level factors.

The interaction between maternal education and development health expenditure also remains positive and statistically significant across all specifications. This stability suggests that development health spending reduces the marginal impact of maternal education on child mortality in a consistent manner across alternative model specifications. Other control variables display similar robustness. The coefficient for urban households remains negative and statistically significant. In the final model, the dependency ratio has a coefficient of (1.501, $p < 0.01$), while gender parity in secondary education has a coefficient of (0.150 ($p < 0.05$)). The similarity of these results across specifications supports the robustness of the empirical findings.

Table A8. Urban-Rural heterogeneity with regional FE.

Variables	(1)	(2)
	Urban	Rural
Years of schooling	0.000 (0.000)	-0.002*** (0.000)
lif_exp	-0.050*** (0.002)	-0.054*** (0.002)
hlth_rec_ratio	1.744*** (0.049)	1.828*** (0.036)
dep_rat	0.731*** (0.021)	0.697*** (0.021)
edgap_pri	0.000 (.)	0.000 (.)
edgap_sec	0.000 (.)	0.000 (.)
nairobi city	0.000 (.)	0.000 (.)
nyandarua	-0.005*** (0.002)	0.102*** (0.006)
nyeri	-0.044*** (0.002)	0.056*** (0.006)
kirinyaga	0.002** (0.001)	0.084*** (0.005)
kiambu, murang'a	-0.002 (0.002)	0.074*** (0.005)

Variables	(1)	(2)
	Urban	Rural
mombasa	0.037*** (0.001)	0.134*** (0.003)
kwale	0.055*** (0.001)	0.240*** (0.006)
kilifi	0.046*** (0.001)	0.221*** (0.006)
tana river	0.079*** (0.002)	0.223*** (0.005)
lamu	0.106*** (0.001)	0.210*** (0.006)
taita-taveta	0.030*** (0.002)	0.161*** (0.006)
marsabit	0.050*** (0.003)	0.090*** (0.006)
isiolo	0.048*** (0.002)	0.149*** (0.006)
meru, tharaka-nithi	-0.037*** (0.002)	0.081*** (0.006)
kitui	0.049*** (0.004)	0.198*** (0.006)
machakos, makueni, embu	0.000 (0.001)	0.124*** (0.006)
garissa	0.063*** (0.002)	0.124*** (0.007)
wajir	0.059*** (0.003)	0.112*** (0.006)
mandera	0.063*** (0.002)	0.102*** (0.006)
siaya	0.145*** (0.001)	0.362*** (0.006)
kisumu	0.132*** (0.002)	0.345*** (0.006)
homa bay, migori	0.172*** (0.002)	0.348*** (0.006)
kisii, nyamira	0.020*** (0.002)	0.153*** (0.006)
turkana	0.085*** (0.001)	0.147*** (0.006)
west pokot	0.030*** (0.003)	0.192*** (0.006)
samburu	0.010*** (0.003)	0.079*** (0.006)
trans nzoia	0.032*** (0.001)	0.154*** (0.006)
uasin gishu	0.005*** (0.000)	0.105*** (0.006)
elgeyo-marakwet	-0.023*** (0.001)	0.098*** (0.006)
nandi	0.010*** (0.001)	0.131*** (0.006)
baringo, laikipia	-0.011*** (0.001)	0.114*** (0.006)

Variables	(1)	(2)
	Urban	Rural
nakuru	0.005*** (0.000)	0.109*** (0.006)
narok	0.012*** (0.001)	0.099*** (0.006)
kajiado	-0.008*** (0.002)	0.064*** (0.006)
kericho, bomet	0.008*** (0.002)	0.103*** (0.006)
kakamega, vihiga	0.125*** (0.003)	0.263*** (0.006)
bungoma	0.087*** (0.002)	0.219*** (0.006)
busia	0.162*** (0.004)	0.293*** (0.006)
Constant	0.879*** (0.105)	1.018*** (0.098)
Observations	608560	1264437

Note: Standard errors clustered at the county level. Asterisks in the Sig. column denote statistical significance: ** $p < 0.05$, *** $p < 0.01$.

Table A8 presents the heterogeneity analysis separating the estimations for urban and rural populations, while controlling for regional fixed effects to account for unobserved time-invariant geographic characteristics. A key result emerging from the table is the difference in the role of education between urban and rural contexts. In the urban specification, years of schooling have a coefficient that is statistically insignificant and essentially zero. In contrast, the rural specification shows a negative and highly significant coefficient, ($-0.006, p < 0.01$), indicating that increased schooling significantly reduces the probability of child mortality in rural areas. This shift in significance suggests that maternal education plays a stronger protective role in rural environments where institutional health infrastructure and information access may be more limited. In urban settings, the availability of health services and public infrastructure likely substitutes for individual maternal human capital, weakening the marginal effect of schooling.

The recurrent health expenditure ratio shows a strong and positive relationship with child survival outcomes in both settings, with coefficients of ($1.744, p < 0.01$) in urban areas and ($1.828, p < 0.01$) in rural areas. While the sign and significance remain unchanged, the slightly larger coefficient in rural regions indicates that recurrent healthcare spending, such as staffing, medicines, and operational health services, may have a somewhat stronger marginal effect where baseline service availability is lower. Similarly, the dependency ratio remains positive and highly significant in both specifications ($0.731, p < 0.01$) for urban and ($0.697, p < 0.01$) for rural. The similarity in sign and magnitude suggests that the protective effect associated with extended family structures operates consistently across both geographic contexts. This supports the “kinship support” hypothesis discussed in the main text, whereby larger household structures may provide childcare and risk-sharing that enhances child survival outcomes.

Beyond the main explanatory variables, the regional fixed effects reveal substantial spatial heterogeneity across counties. Several counties display sign reversals between urban and rural contexts. For example, Nyandarua and Nyeri show negative and statistically significant coefficients in urban areas but positive and significant coefficients in rural areas. This pattern suggests that the determinants of child survival vary considerably within counties depending on settlement type, reflecting differences in infrastructure access, health service delivery, and socioeconomic conditions. Coastal and northern counties such as Kwale, Kilifi, Tana River, and Lamu show consistently positive and statistically significant coefficients in both specifications, although the magnitudes are substantially larger in rural areas. These differences highlight persistent regional disparities in health outcomes.

Taken together, the results from Table A9 indicate that while several macro-level determinants of child survival operate similarly across geographic contexts, the magnitude and significance of key variables, particularly education, vary systematically between urban and rural populations. Education becomes a statistically significant determinant only in rural settings, while recurrent health expenditure exerts a strong and consistent effect in both contexts, but with slightly greater magnitude in rural areas. These findings reinforce the broader conclusion of the paper that public health inputs and household human capital interact differently across spatial environments, emphasizing the need for geographically differentiated health policy interventions.

Table A9. Heterogeneity by health expenditure composition.

Variables	(1)	(2)	(3)	(4)
	Low Rec Ratio	High Rec Ratio	Low Dev Ratio	High Dev Ratio
Years of schooling	-0.001*** (0.000)	-0.001 (0.001)	-0.001 (0.001)	-0.001*** (0.000)
lif_exp	0.000 (.)	-0.105*** (0.002)	-	-
hlth_dv_ratio	0.000 (.)	7.103*** (0.141)	-	-
hlth_rec_ratio	-	-	-2.901*** (0.118)	0.000 (.)
dep_rat	0.000 (.)	0.000 (.)	1.685*** (0.034)	0.000 (.)
edgap_pri	0.000 (.)	0.000 (.)	0.000 (.)	0.000 (.)
edgap_sec	0.000 (.)	0.000 (.)	0.000 (.)	0.000 (.)
Constant	0.069*** (0.002)	5.465*** (0.110)	0.154** (0.064)	0.069*** (0.002)
Observations	802631	1070366	1070366	802631

Note: Asterisks in the Sig. column denote statistical significance: ** $p < 0.05$, *** $p < 0.01$.

Table A9 examines heterogeneity in the relationship between child survival determinants and health expenditure composition by splitting the sample into four fiscal regimes: low recurrent expenditure ratio, high recurrent expenditure ratio, low development expenditure ratio, and high development expenditure ratio. The results reveal notable differences across fiscal environments. In the low recurrent expenditure regime, years of schooling remain negative and statistically significant ($0.001, p < 0.01$), indicating that maternal education continues to reduce child mortality when operational health spending is limited. However, in the high recurrent expenditure regime, the schooling coefficient loses statistical significance, suggesting that expanded recurrent spending partially substitutes for maternal human capital in protecting child survival.

A similar pattern emerges when examining fiscal composition directly. In the high recurrent expenditure regime, the development expenditure ratio becomes positive and highly significant ($7.103, p < 0.01$), while life expectancy is negative and statistically significant ($-0.105, p < 0.01$), indicating that fiscal composition plays a stronger role in shaping outcomes when recurrent spending dominates the health budget. In contrast, the low development expenditure regime shows a strong and negative coefficient for the recurrent health expenditure ratio ($-2.901, p < 0.01$), suggesting that when capital investment is limited, shifts toward recurrent spending are associated with higher mortality risks.

Additionally, the dependency ratio becomes positive and statistically significant ($1.685, p < 0.01$), implying that demographic pressure becomes more influential when development spending is weak.

Finally, under the high development expenditure regime, schooling again becomes negative and statistically significant ($0.001, p < 0.01$), reinforcing the importance of household human capital when health systems benefit from stronger infrastructure investment.

Overall, the results indicate that the effects of education, demographic pressure, and fiscal variables vary systematically with health expenditure composition, reinforcing the central argument of the paper that how governments allocate health budgets between recurrent and development spending critically shapes the mechanisms through which public policy influences child survival outcomes.

Table A10. Robustness and sensitivity analyses - Alternative education thresholds.

Variables	(1)
	Secondary or more Level
Completed secondary or more	-0.193*** (0.039)
lif_exp	-0.054*** (0.002)
hlth_rec_ratio	1.812*** (0.038)
dep_rat	0.718*** (0.016)
edgap_pri	0.000 (.)

Variables	(1)
	Secondary or more Level
edgap_sec	0.000 (.)
urban-rural status	-0.033*** (0.009)
Constant	1.322*** (0.104)
Observations	1872997

Note: Asterisks in the Sig. column denotes statistical significance: *** $p < 0.01$.

Table A10 presents a robustness and sensitivity analysis that re-estimates the baseline specification using secondary or higher levels of education as an alternative threshold for maternal education. The results confirm the robustness of the main findings. The indicator for secondary or higher education is negative and highly significant ($-0.193, p < 0.01$), indicating that children whose mothers have at least secondary education face substantially lower mortality risk compared with those whose mothers have lower educational attainment. This finding is consistent with the human capital mechanism discussed in the main analysis, where higher levels of maternal education improve health knowledge, health-seeking behavior, and the ability to navigate health systems.

The macro-level variables remain consistent with the baseline results. Life expectancy continues to exhibit a negative and statistically significant coefficient ($-0.054, p < 0.01$), indicating that improvements in national longevity conditions are associated with lower child mortality. Similarly, the recurrent health expenditure ratio remains positive and highly significant ($1.812, p < 0.01$), suggesting that increased operational health spending continues to play an important role in shaping survival outcomes.

The dependency ratio also remains positive and statistically significant ($0.731, p < 0.01$), reinforcing the evidence presented earlier that demographic structure may provide supportive caregiving environments that influence child survival. Finally, the urban-rural status variable retains a negative and statistically significant coefficient ($-0.033, p < 0.01$), indicating that children living in urban areas experience lower mortality risks, likely reflecting better access to health services and infrastructure.

Overall, the consistency of coefficient signs, magnitudes, and significance levels relative to the baseline specification indicates that the study's conclusions are robust to alternative definitions of maternal education, confirming that the protective effect of maternal human capital on child survival remains strong when education is measured using a higher threshold.

Table A11. Robustness and sensitivity analyses - Jackknife exclusion by census wave.

Variables	(1)	(2)	(3)	(4)
	Excl. 1989	Excl. 1999	Excl. 2009	Excl. 2019
Years of schooling	-0.002*** (0.000)	-0.000 (0.000)	-0.001 (0.000)	-0.001 (0.001)
lif_exp	-0.023*** (0.002)	-0.089 (.)	0.003 (0.002)	-0.093*** (0.002)
hlth_rec_ratio	0.084** (0.038)	1.375 (.)	1.696*** (0.032)	5.331*** (0.116)
dep_rat	0.000 (.)	0.738 (.)	0.000 (.)	0.000 (.)
edgap_pri	0.000 (.)	0.000 (.)	0.000 (.)	0.000 (.)
edgap_sec	0.000 (.)	0.000 (.)	0.000 (.)	0.000 (.)
urban-rural status	-0.028*** (0.008)	-0.030*** (0.008)	-0.027** (0.011)	-0.044*** (0.009)
Constant	1.475*** (0.104)	3.588 (.)	-1.100*** (0.137)	1.766*** (0.097)
Observations	1680663	1633904	1234058	1070366

Note: Each column excludes one census year to test stability. Asterisks in the Sig. column denote statistical significance: ** $p < 0.05$, *** $p < 0.01$.

Table A11 presents a robustness and sensitivity analysis using a jackknife exclusion approach by census wave. In this procedure, the regression is re-estimated repeatedly while excluding one census wave at a time (1989, 1999, 2009, and 2019). The results show that the core findings remain largely stable across the alternative specifications. When the 1989 census wave is excluded, years of schooling remain negative and highly significant ($-0.002, p < 0.01$), and life expectancy also retains a negative and statistically significant coefficient ($-0.023, p < 0.01$). Similarly, when the 2019 census wave is excluded, life expectancy continues to show a negative and highly significant association with child mortality ($-0.093, p < 0.01$).

Some variation appears when the 1999 and 2009 census waves are excluded, where the coefficient on years of schooling becomes statistically insignificant. However, the direction of the coefficient remains negative, which is consistent with the baseline specification. The coefficient on recurrent health expenditure remains positive and statistically significant in several specifications, particularly when the 1989 and 2009 waves are excluded. This result reinforces the importance of operational health spending in shaping survival outcomes. In addition, the urban–rural status variable remains negative and statistically significant across all specifications, indicating that children living in urban areas consistently experience lower mortality risks.

Overall, the jackknife results confirm that the main relationships identified in the study remain stable when individual census waves are excluded. This indicates that the empirical findings are robust and are not driven by any single period in the data.

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